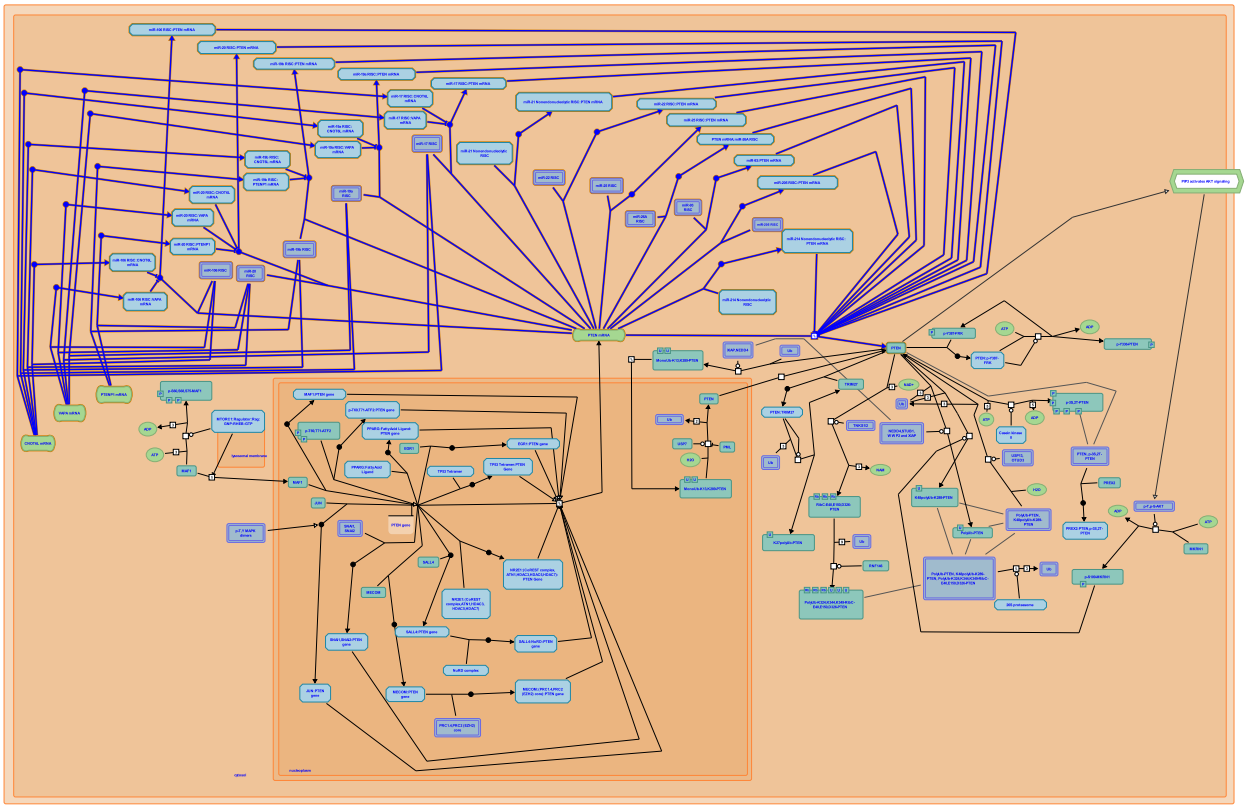
Regulation of PTEN mRNA translation



Carracedo, A., Kriplani, N., Leslie, N., Matthews, L., Orlic-Milacic, M., Salmena, L., Thorpe, L., Yuzugullu, H., Zhao, JJ.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the Reactome Textbook.

03/05/2024

Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.
The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

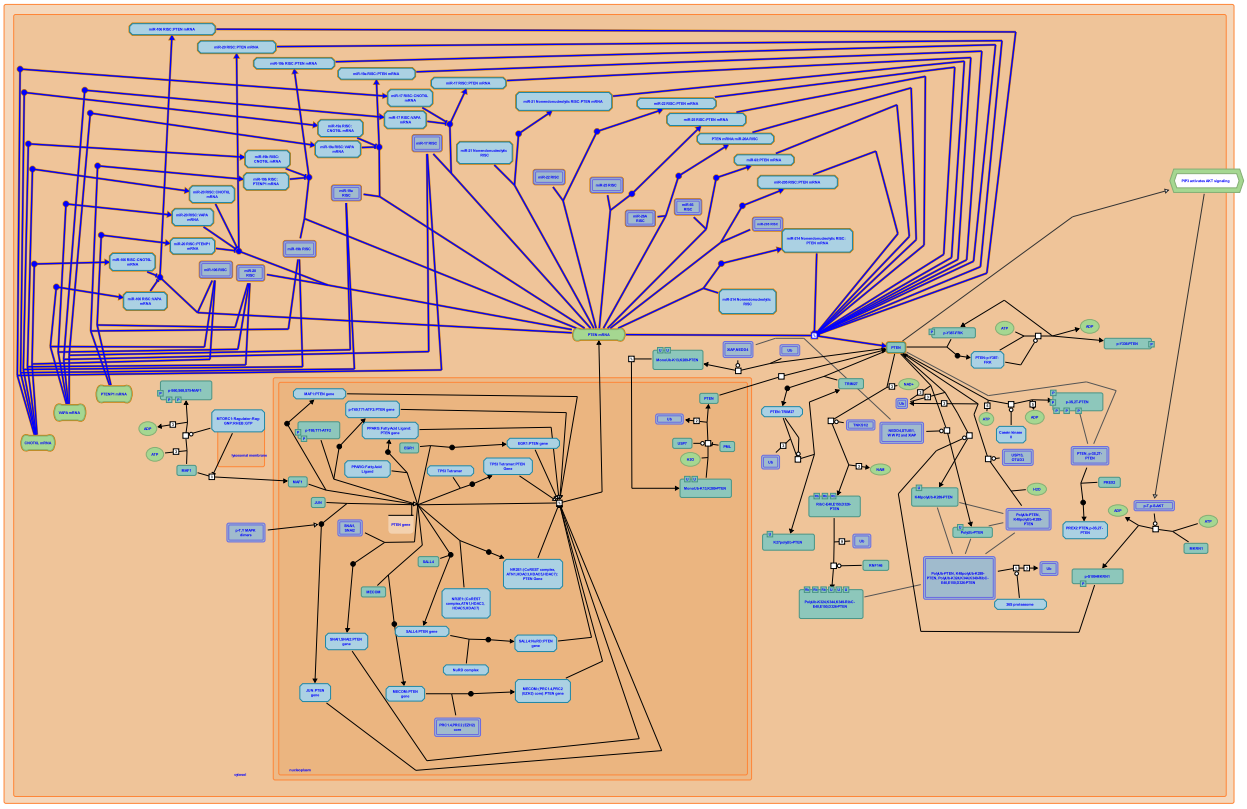
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

Regulation of PTEN mRNA translation ↗

Stable identifier: R-HSA-8943723



MicroRNAs miR-17, miR-19a, miR-19b, miR-20a, miR-20b, miR-21, miR-22, miR-25, miR 26A1, miR 26A2, miR-93, miR-106a, miR-106b, miR 205, and miR 214 and bind PTEN mRNA and inhibit its translation into protein. These microRNAs are altered in cancer and can account for changes in PTEN levels. There is evidence that PTEN mRNA translation is also inhibited by other microRNAs, such as miR-302 and miR-26B, and these microRNAs will be annotated when additional experimental details become available (Meng et al. 2007, Xiao et al. 2008, Yang et al. 2008, Huse et al. 2009, Kim et al. 2010, Poliseno, Salmena, Riccardi et al. 2010, Zhang et al. 2010, Tay et al. 2011, Qu et al. 2012, Cai et al. 2013). In addition, coding and non coding RNAs can prevent microRNAs from binding to PTEN mRNA. These RNAs are termed competing endogenous RNAs or ceRNAs. Transcripts of the pseudogene PTENP1 and mRNAs transcribed from SERINC1, VAPA and CNOT6L genes exhibit this activity (Poliseno, Salmena, Zhang et al. 2010, Tay et al. 2011, Tay et al. 2014).

Literature references

Cai, J., Huang, Y., Li, R., Yang, Y., Yuan, J., Fang, L. et al. (2013). miR-205 targets PTEN and PHLPP2 to augment AKT signaling and drive malignant phenotypes in non-small cell lung cancer. *Cancer Res., 73*, 5402-15. ↗

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Kong, W., O'Donnell, JD., Kruk, PA., Zhao, JJ., He, L., Wenham, RM. et al. (2008). MicroRNA expression profiling in human ovarian cancer: miR-214 induces cell survival and cisplatin resistance by targeting PTEN. *Cancer Res., 68*, 425-33. ↗

Weiss, D., Karreth, F., Tan, SM., Di Cunto, F., Rigoutsos, I., Provero, P. et al. (2011). Coding-independent regulation of the tumor suppressor PTEN by competing endogenous mRNAs. *Cell, 147*, 344-57. ↗

Park, PJ., Johnson, MD., Jiang, X., Kim, H., Huang, W., Pennicooke, B. (2010). Integrative genome analysis reveals an oncomir/oncogene cluster regulating glioblastoma survivorship. *Proc. Natl. Acad. Sci. U.S.A., 107*, 2183-8. ↗

Editions

2016-08-11	Authored	Carracedo, A., Salmena, L.
2016-09-30	Reviewed	Leslie, N., Kriplani, N.
2016-10-28	Authored	Orlic-Milacic, M.
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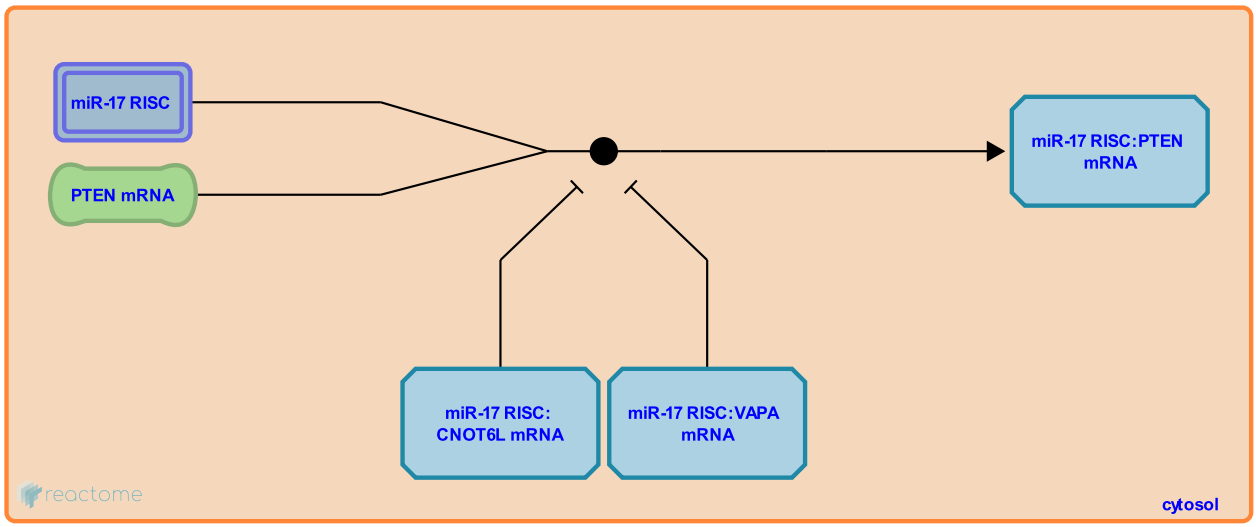
miR-17 microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944483

Type: binding

Compartments: cytosol



MicroRNA miR-17-5p, one of the two mature products of miR-17, binds the 3'UTR of PTEN mRNA (Xiao et al. 2008, Poliseno et al. 2010). miR-17 causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-17 also functions as a part of the nonendonucleolytic RISC.

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Zhang, B., Xiao, C., Kutok, JL., Henderson, JM., Wang, J., Calado, DP. et al. (2008). Lymphoproliferative disease and autoimmunity in mice with increased miR-17-92 expression in lymphocytes. *Nat. Immunol., 9*, 405-14. ↗

Editions

2016-08-11	Authored	Carracedo, A., Salmena, L.
2016-09-30	Reviewed	Leslie, N., Kriplani, N.
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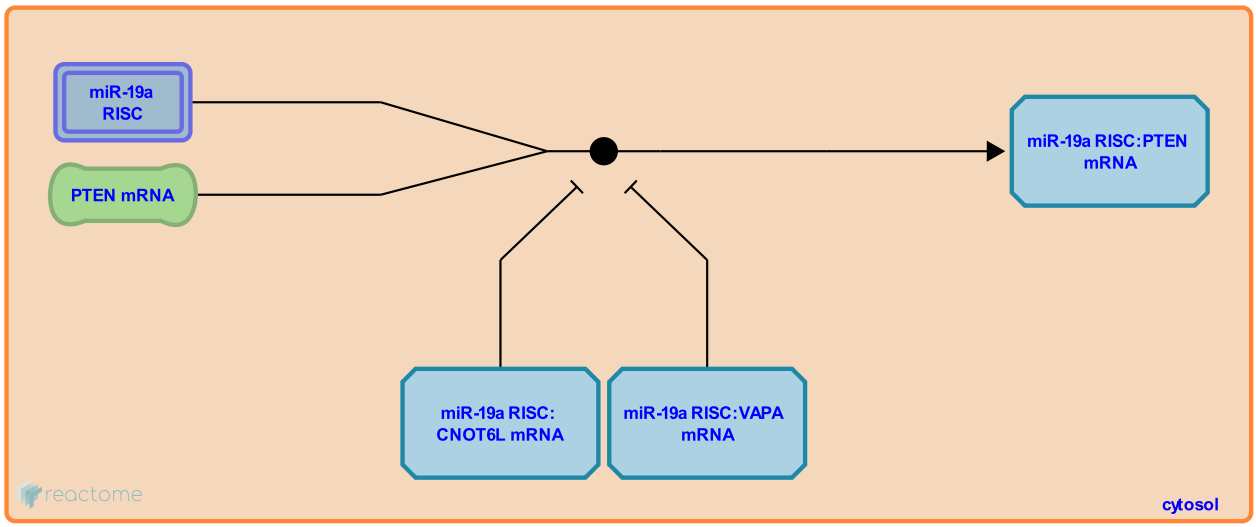
miR-19a microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944522

Type: binding

Compartments: cytosol



MicroRNA miR-19a-3p, one of the two mature products of miR-19a, binds the 3'UTR of PTEN mRNA (Xiao et al. 2008, Poliseno, Salmena, Riccardi et al. 2010). miR-19a microRNA causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-19a microRNA also functions as a part of the nonendonucleolytic RISC.

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Zhang, B., Xiao, C., Kutok, JL., Henderson, JM., Wang, J., Calado, DP. et al. (2008). Lymphoproliferative disease and autoimmunity in mice with increased miR-17-92 expression in lymphocytes. *Nat. Immunol., 9*, 405-14. ↗

Editions

2016-09-30	Reviewed	Leslie, N., Kriplani, N.
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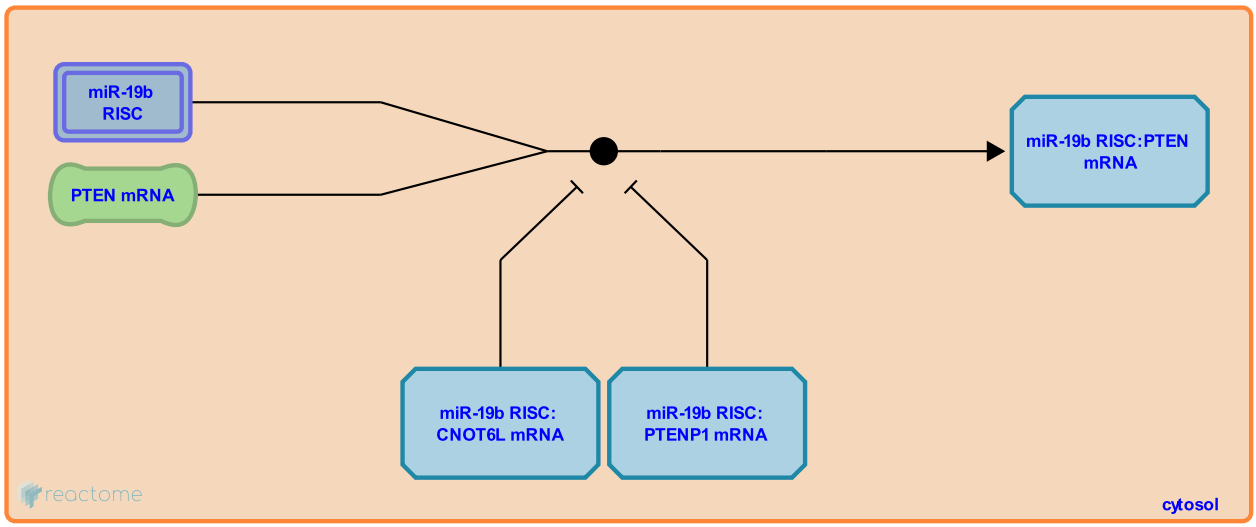
miR-19b microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8948569

Type: binding

Compartments: cytosol



MicroRNA miR-19b, encoded by two genomic loci, MIR19B1 and MIR19B2, is homologous to miR-19a and also binds to the 3'UTR of PTEN mRNA (Polseno, Salmena, Zhang et al. 2010). miR-19b microRNA causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-19b microRNA also functions as a part of the nonendonucleolytic RISC.

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Pandolfi, PP., Carver, B., Haveman, WJ., Poliseno, L., Zhang, J., Salmena, L. (2010). A coding-independent function of gene and pseudogene mRNAs regulates tumour biology. *Nature, 465*, 1033-8. ↗

Editions

2016-09-30	Reviewed	Leslie, N., Kriplani, N.
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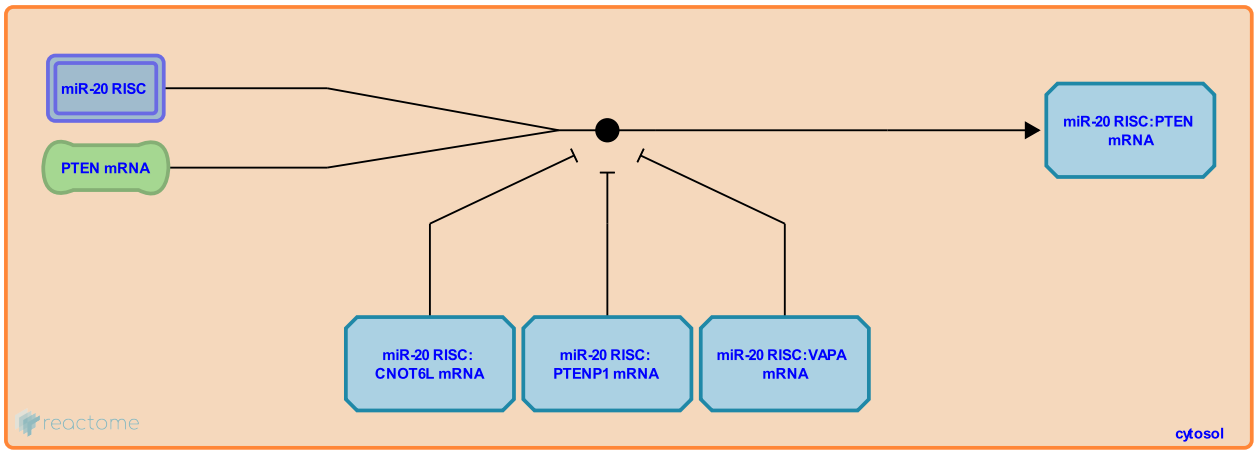
miR-20 microRNAs bind PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8945709

Type: binding

Compartments: cytosol



MicroRNAs miR-20a-5p and miR-20b-5p, encoded by MIR20A and MIR20B genes, respectively, bind the 3'UTR of PTEN mRNA and downregulate PTEN mRNA translation (Poliseno et al. 2010, Tay et al. 2011). miR-20a-5p downregulates both PTEN mRNA and protein levels and is thus considered to function as part of the endonucleolytic RISC, but it may also function as a part of nonendonucleolytic RISC (Poliseno et al. 2010). miR-20b presumably functions in a similar manner (Tay et al. 2011).

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Weiss, D., Karreth, F., Tan, SM., Di Cunto, F., Rigoutsos, I., Provero, P. et al. (2011). Coding-independent regulation of the tumor suppressor PTEN by competing endogenous mRNAs. *Cell, 147*, 344-57. ↗

Pandolfi, PP., Carver, B., Haveman, WJ., Poliseno, L., Zhang, J., Salmena, L. (2010). A coding-independent function of gene and pseudogene mRNAs regulates tumour biology. *Nature, 465*, 1033-8. ↗

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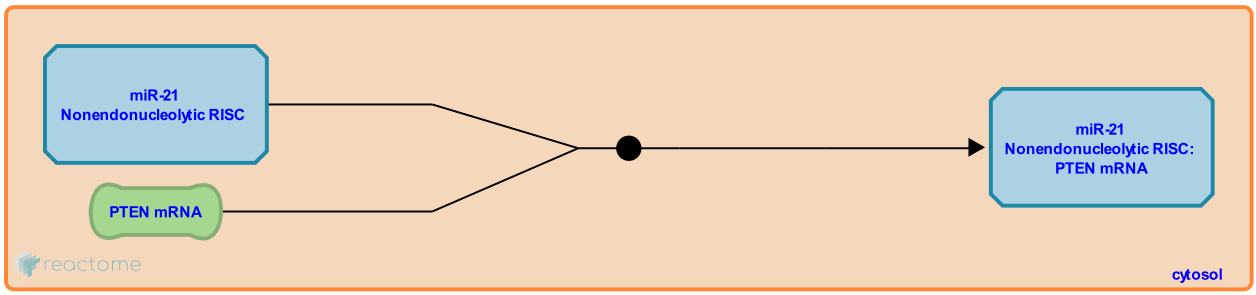
miR-21 nonendonucleolytic RISC binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944706

Type: binding

Compartments: cytosol



miR-21-5p, one of the two mature products of miR-21, binds the 3'UTR of PTEN mRNA to inhibit PTEN mRNA translation. miR-21 only affects PTEN protein level and thus presumably functions as part of the nonendonucleolytic RISC (Meng et al. 2007, Zhang et al. 2010)

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Yang, GH., Liu, Q., Wang, JJ., Zhang, JG., Jiang, K., Zhao, F. (2010). MicroRNA-21 (miR-21) represses tumor suppressor PTEN and promotes growth and invasion in non-small cell lung cancer (NSCLC). *Clin. Chim. Acta, 411*, 846-52. ↗

Wehbe-Janek, H., Patel, T., Meng, F., Henson, R., Jacob, ST., Ghoshal, K. (2007). MicroRNA-21 regulates expression of the PTEN tumor suppressor gene in human hepatocellular cancer. *Gastroenterology, 133*, 647-58. ↗

Editions

2016-08-11	Authored	Carracedo, A., Salmena, L.
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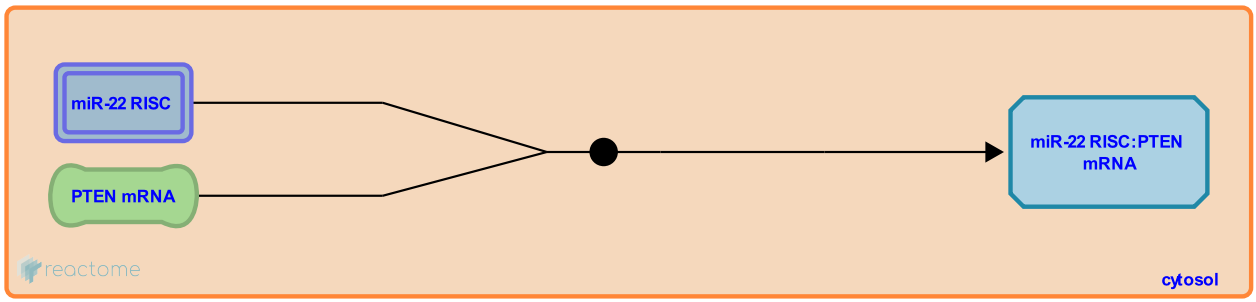
miR-22 microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944538

Type: binding

Compartments: cytosol



MicroRNA miR-22-3p, one of the two mature products of miR-22, binds the 3'UTR of PTEN mRNA (Poliseno et al. 2010). miR-22 causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-22 also functions as a part of the nonendonucleolytic RISC.

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Editions

2016-09-30	Reviewed	Leslie, N., Kriplani, N.
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2017-05-09	Edited	Orlic-Milacic, M.

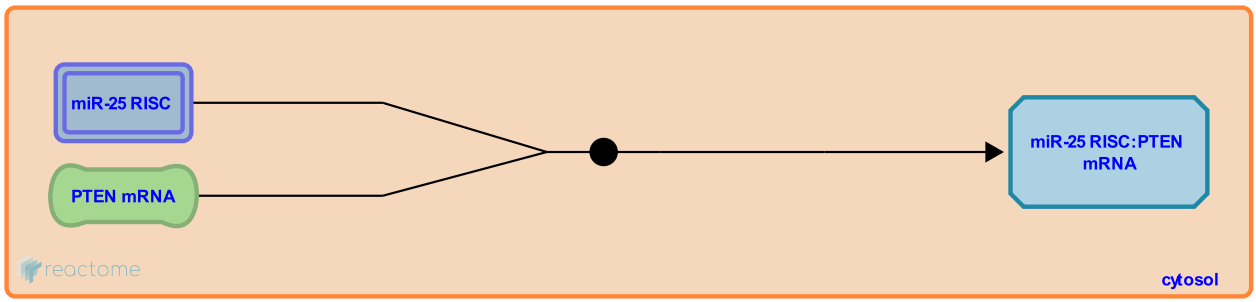
miR-25 microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944569

Type: binding

Compartments: cytosol



MicroRNA miR-25-3p, one of the two mature products of miR-25, binds the 3'UTR of PTEN mRNA (Poliseno et al. 2010). miR-25 causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-25 also functions as a part of the nonendonucleolytic RISC.

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Editions

2016-09-30	Reviewed	Leslie, N., Kriplani, N.
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2017-05-09	Edited	Orlic-Milacic, M.

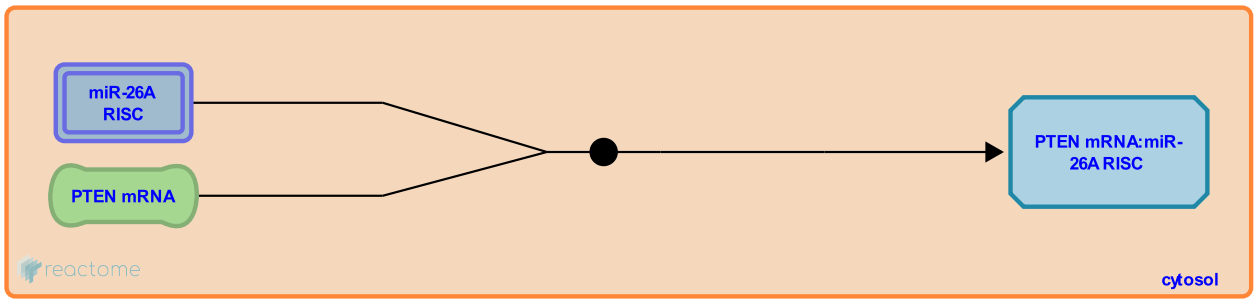
miR-26A microRNAs bind PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-2318752

Type: binding

Compartments: cytosol



MIR26A microRNAs, miR-26A1 and miR-26A2, transcribed from genes on chromosome 3 and 12, respectively, bind PTEN mRNA (Huse et al. 2009).

The MIR26A2 locus is frequently amplified in glioma tumors that retain one wild-type PTEN allele. The resulting miR-26A2 overexpression leads to down-regulation of PTEN protein level. Overexpression of miR-26A2 was shown to enhance tumorigenesis and negatively correlates with the loss of heterozygosity at the PTEN locus in a mouse PTEN +/- glioma model, based on monoallelic PTEN loss (Huse et al. 2009, Kim et al. 2010).

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Park, PJ., Johnson, MD., Jiang, X., Kim, H., Huang, W., Pennicooke, B. (2010). Integrative genome analysis reveals an oncomir/oncogene cluster regulating glioblastoma survivorship. *Proc. Natl. Acad. Sci. U.S.A., 107*, 2183-8. ↗

Rouhanifard, SH., le Sage, C., Hambardzumyan, D., Brennan, C., Holland, EC., Sohn-Lee, C. et al. (2009). The PTEN-regulating microRNA miR-26a is amplified in high-grade glioma and facilitates gliomagenesis in vivo. *Genes Dev., 23*, 1327-37. ↗

Editions

2012-07-18	Authored	Orlic-Milacic, M.
2012-08-03	Edited	Matthews, L.
2012-08-13	Reviewed	Zhao, JJ., Yuzugullu, H., Thorpe, L.
2016-08-11	Reviewed	Carracedo, A., Salmena, L.
2016-09-30	Reviewed	Leslie, N., Kriplani, N.

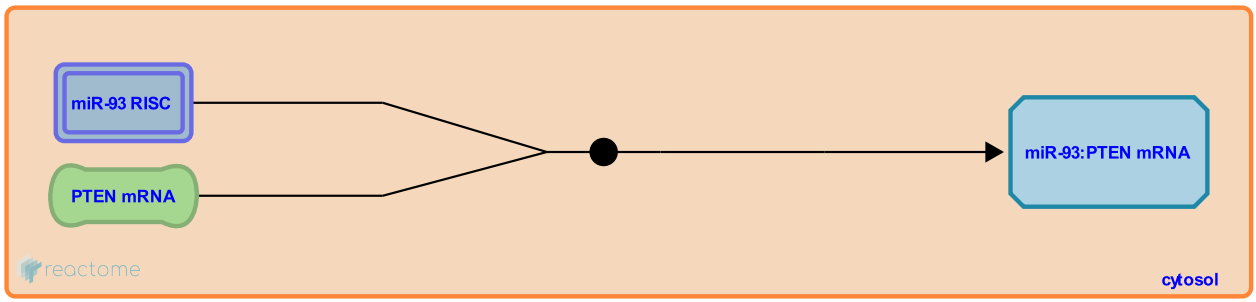
miR-93 microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944599

Type: binding

Compartments: cytosol



MicroRNA miR-93-5p, one of the two mature products of miR-93, binds the 3'UTR of PTEN mRNA (Poliseno et al. 2010). miR-93 causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-93 also functions as a part of the nonendonucleolytic RISC.

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Editions

2016-09-30	Reviewed	Leslie, N., Kriplani, N.
2016-11-03	Authored	Orlic-Milacic, M.
2017-05-09	Edited	Orlic-Milacic, M.

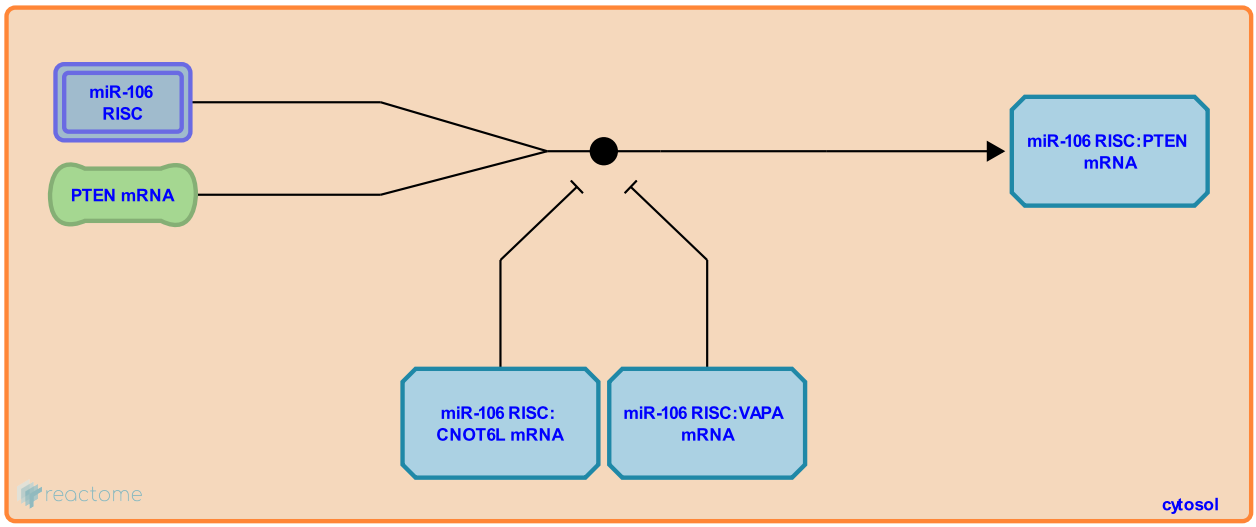
miR-106 microRNAs bind PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944632

Type: binding

Compartments: cytosol



MicroRNA miR-106b-5p, one of the two mature products of miR-106b, binds the 3'UTR of PTEN mRNA (Poliseno et al. 2010). miR-106b causes reduction in both PTEN mRNA and protein levels and is thus shown to function as a part of the endonucleolytic RISC. It is possible that miR-106b also functions as a part of the nonendonucleolytic RISC. MicroRNA miR-106a-5p, one of the two mature products of miR-106a, is homologous to miR-106b and binds to the 3'UTR of PTEN mRNA. miR-106a presumably functions in a manner similar to miR-106b (Tay et al. 2011).

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Rameh, L., Varmeh, S., Sportoletti, P., Fornari, A., Loda, M., Poliseno, L. et al. (2010). Identification of the miR-106b~25 microRNA cluster as a proto-oncogenic PTEN-targeting intron that cooperates with its host gene MCM7 in transformation. *Sci Signal, 3*, ra29. ↗

Weiss, D., Karreth, F., Tan, SM., Di Cunto, F., Rigoutsos, I., Provero, P. et al. (2011). Coding-independent regulation of the tumor suppressor PTEN by competing endogenous mRNAs. *Cell, 147*, 344-57. ↗

Editions

2016-08-11	Authored	Carracedo, A., Salmena, L.
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2017-05-09	Edited	Orlic-Milacic, M.

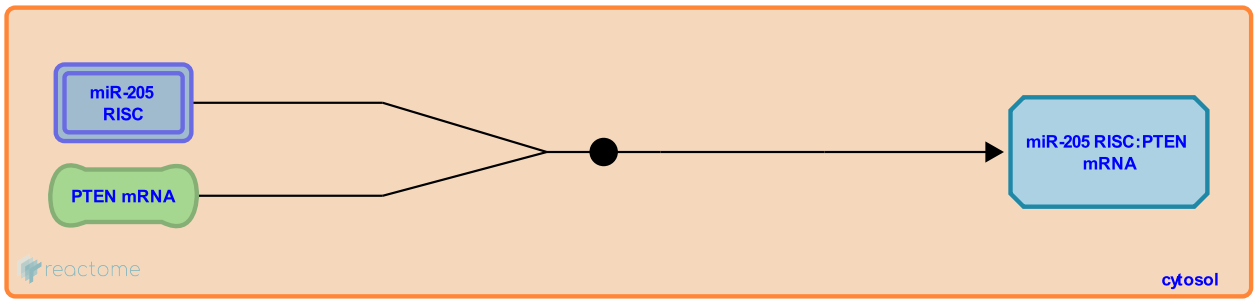
miR-205 microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944684

Type: binding

Compartments: cytosol



MicroRNA miR-205-5p, one of the two mature products of miR-205, binds the 3'UTR of the PTEN mRNA, resulting in downregulation of PTEN mRNA and protein levels. miR-205 functions as part of both endonucleolytic and nonendonucleolytic RISCs (Qu et al. 2012, Cai et al. 2013). In addition to PTEN, miR-205 targets another negative regulator of PI3K/AKT signaling - the protein serine/threonine phosphatase PHLPP2 (Cai et al. 2013).

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Cai, J., Huang, Y., Li, R., Yang, Y., Yuan, J., Fang, L. et al. (2013). miR-205 targets PTEN and PHLPP2 to augment AKT signaling and drive malignant phenotypes in non-small cell lung cancer. *Cancer Res., 73*, 5402-15. ↗

Liang, Z., Zhang, R., Huang, J., Wang, X., Su, C., Qu, C. et al. (2012). MiR-205 determines the radioresistance of human nasopharyngeal carcinoma by directly targeting PTEN. *Cell Cycle, 11*, 785-96. ↗

Editions

2016-08-11	Authored	Carracedo, A., Salmena, L.
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2017-05-09	Edited	Orlic-Milacic, M.

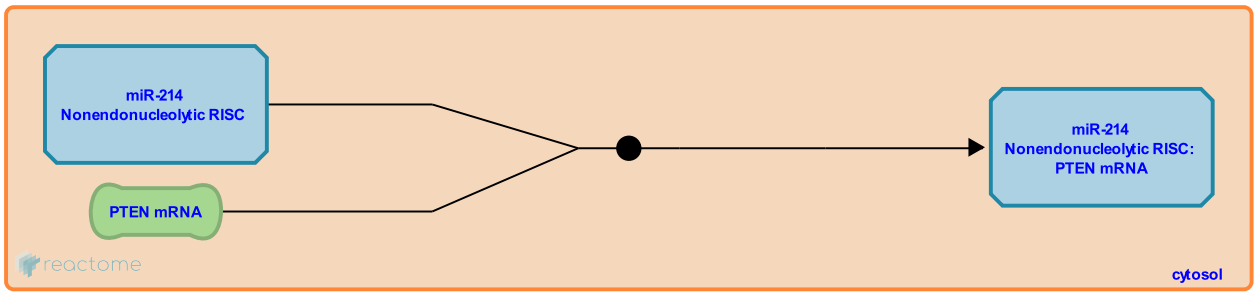
miR-214 microRNA binds PTEN mRNA ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944650

Type: binding

Compartments: cytosol



MicroRNA miR-214-3p, one of the two mature products of miR-214, binds to the 3'UTR of the PTEN mRNA and inhibits PTEN mRNA translation. As miR-214 reduces PTEN protein levels but not PTEN mRNA levels, miR-214 presumably functions as part of the nonendonucleolytic RISC. miR-214 is frequently overexpressed in ovarian cancer (Yang et al. 2008).

Followed by: PTEN mRNA translation is negatively regulated by microRNAs

Literature references

Kong, W., O'Donnell, JD., Kruk, PA., Zhao, JJ., He, L., Wenham, RM. et al. (2008). MicroRNA expression profiling in human ovarian cancer: miR-214 induces cell survival and cisplatin resistance by targeting PTEN. *Cancer Res., 68*, 425-33. ↗

Editions

2016-08-11	Authored	Carracedo, A., Salmena, L.
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2016-11-03	Authored	Orlic-Milacic, M.
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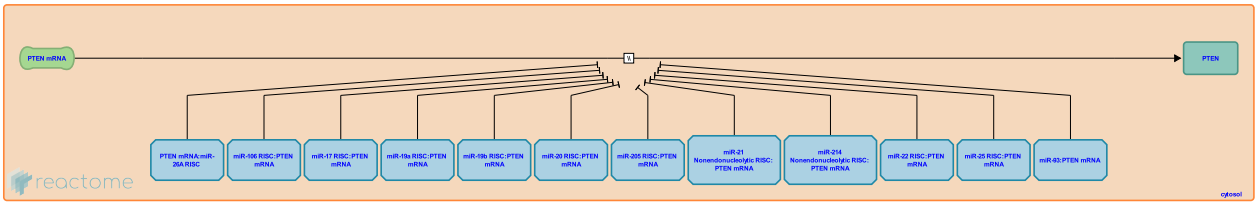
PTEN mRNA translation is negatively regulated by microRNAs ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8944497

Type: omitted

Compartments: cytosol



PTEN protein synthesis is negatively regulated by microRNAs miR-26A1 and miR-26A2, which recruit the RISC complex to PTEN mRNA. Overexpression of miR-26A2, caused by genomic amplification of MIR26A2 locus on chromosome 12, is frequently observed in human brain glioma tumors possessing one wild-type PTEN allele, and is thought to contribute to tumor progression by repressing PTEN protein expression from the remaining allele (Huse et al. 2009). Other microRNAs, which may also be altered in cancer, such as miR-17, miR-19a, miR-19b, miR-20a, miR-20b, miR-21, miR-22, miR-25, miR-93, miR-106a, miR-106b, miR 205, and miR 214, also bind PTEN mRNA and inhibit its translation into protein (Meng et al. 2007, Xiao et al. 2008, Yang et al. 2008, Kim et al. 2010, Poliseno, Salmena, Riccardi et al. 2010, Zhang et al. 2010, Tay et al. 2011, Qu et al. 2012, Cai et al. 2013).

Preceded by: miR-106 microRNAs bind PTEN mRNA, miR-19b microRNA binds PTEN mRNA, miR-25 microRNA binds PTEN mRNA, miR-19a microRNA binds PTEN mRNA, miR-22 microRNA binds PTEN mRNA, miR-214 microRNA binds PTEN mRNA, miR-26A microRNAs bind PTEN mRNA, miR-205 microRNA binds PTEN mRNA, miR-20 microRNAs bind PTEN mRNA, miR-21 nonendonucleolytic RISC binds PTEN mRNA, miR-17 microRNA binds PTEN mRNA, miR-93 microRNA binds PTEN mRNA

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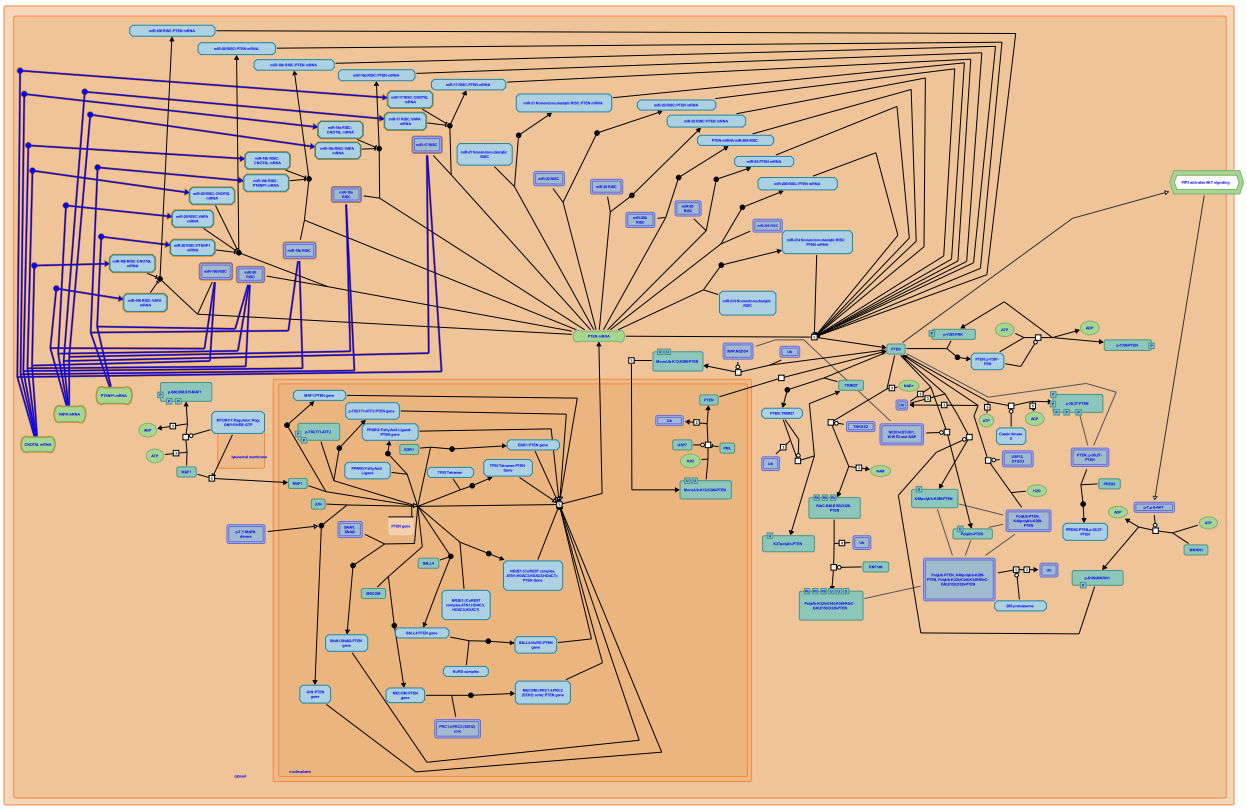
Competing endogenous RNAs (ceRNAs) regulate PTEN translation ↗

Location: Regulation of PTEN mRNA translation

Stable identifier: R-HSA-8948700



Coding and non-coding RNAs can prevent microRNAs from binding to PTEN mRNA. These RNAs are termed competing endogenous RNAs or ceRNAs. Transcripts of the pseudogene PTENP1 and mRNAs transcribed from SERINC1, VAPA and CNOT6L genes exhibit this activity (Poliseno et al. 2010, Tay et al. 2011, Tay et al. 2014). SERINC1 mRNA will be annotated in this context when additional experimental details become available.

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Pandolfi, PP., Carver, B., Haveman, WJ., Poliseno, L., Zhang, J., Salmena, L. (2010). A coding-independent function of gene and pseudogene mRNAs regulates tumour biology. *Nature, 465*, 1033-8. ↗

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